# INTRAUTERINE TREATMENT OF FETAL SUPRAVENTRICULAR TACHYCARDIA COMPLICATED BY FETAL HYDROPS: LIVEBIRTH AT TERM

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## Abstract

***Introduction:*** Fetal tachycardia is a rare complication during pregnancy, with supraventricular tachycardia (SVT) being a common cause of a primary sustained fetal tachyarrhythmia. If developed early in pregnancy, it can lead to non-immune fetal hydrops (FH). Multidisciplinary approach is mandatory between pediatric cardiologists (both paediatric and adult), neonatologist, and maternal-fetal medicine specialist.

***Case Presentation:*** We present a case of fetal SVT, complicated with FH at 24 weeks' gestation, which was successfully treated with transplacental flecainide to emphasize the need for prenatal evaluation of pregnancies complicated by FH and provision of appropriate treatment to optimize outcome. 35-year-old MPLK; with a gravidity of 1 and parity 0, was referred to our Fetal Medicine Clinic due to an increased fetal heart rate (FHR) of 243 beats per minute during a routine fetal anatomical survey scan at 22 weeks gestational age (GA). The pregnant woman and the fetus had no clinical symptoms, and clinical examinations and investigations revealed no organic lesions. The fetus developed congestive heart failure evidenced by cardiomegaly, pericardial effusion, ascites, and skin edema at 24 weeks' gestation. The couple made an informed decision for transplacental antiarrhythmic therapy which resulted in successful fetal cardioversion, with minimal maternal side effects. Delivery was conducted at 37W GA, and baby is currently 4 months old and well.

***Conclusion:*** Prenatal evaluation of pregnancies complicated by fetal hydrops is necessary to determine the underlying etiology and provide appropriate treatment to optimize outcome.

***Keywords:*** *fetal tachycardia, supraventricular tachycardia, fetal hydrops, echocardiography, cardioversion*

## Introduction

Fetal tachycardia (FHR ≥180 bpm) affects 1% of pregnancies, with sustained tachyarrhythmias occurring in 0.1%[1]. Supraventricular tachycardia (SVT) is the most common type[2], and if untreated, can lead to fetal hydrops (FH) and intrauterine death. Diagnosis relies on Doppler ultrasound in resource-limited settings, and management includes monitoring, transplacental therapy, or preterm delivery. This case is unique due to the early diagnosis of SVT with FH at 24 weeks' gestation and its successful resolution with transplacental flecainide, demonstrating the potential for early intervention even in low-resource settings.

## Cases Presentation

A 35-year-old primigravida was referred at 22 weeks' gestation due to fetal tachycardia (FHR: 243 bpm) detected on routine ultrasound. She had no systemic illness, infections, or thyroid dysfunction, and all antenatal labs were normal. Fetal echocardiography confirmed a structurally normal heart with persistent SVT (1:1 atrioventricular conduction) [Fig.1]. At 24 weeks, the tachycardia persisted (244 bpm) with evolving hydrops fetalis (cardiomegaly, pericardial effusion, ascites, and skin edema), leading to a definitive diagnosis of fetal SVT complicated by hydrops [Fig.2a,b]. Maternal transplacental therapy was initiated with oral digoxin, but fetal tachycardia persisted. At 25 weeks, flecainide was added, resulting in a gradual reduction of FHR to 140–160 bpm by 27–31 weeks [Fig.3], with resolution of hydrops [Fig.2c]. At 32 weeks, the mother developed transient headaches and blurred vision, prompting a reduction in flecainide dosage. At 37 weeks, she underwent an elective cesarean section, delivering a healthy male baby (2.6 kg, APGAR 8/9) with a stable heart rate of 120 bpm. The neonate required no antiarrhythmic therapy and was discharged after brief NICU monitoring. At five months, the infant was thriving with normal growth and development. Parents were counseled on potential SVT recurrence and advised on long-term pediatric cardiology follow-up.

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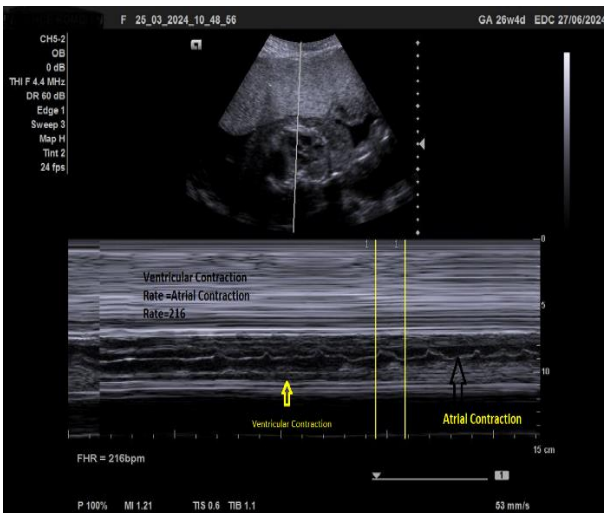


***Figure 1: Fetal Tachyarrythmia***

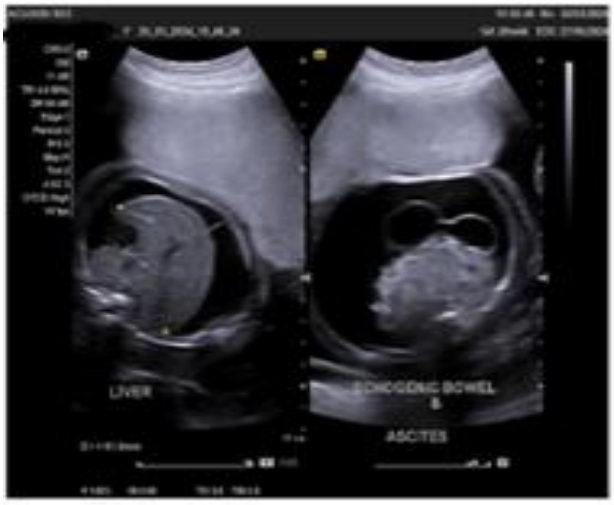


***Figure 2a: Thermal Images at (2a) 26 weeks 4 days showing regression of fetal ascites***

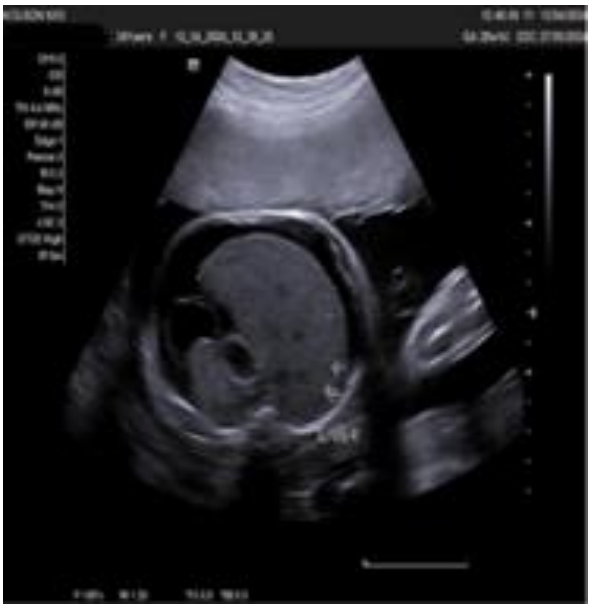

***Figure 2b: Thermal Images 29 weeks days 1 day showing regression of fetal ascites***

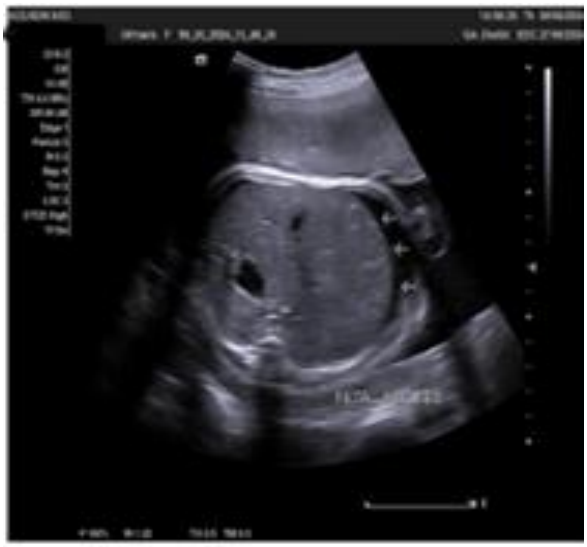

***Figure 2c: 33 weeks 0 days showing regression of fetal ascites***

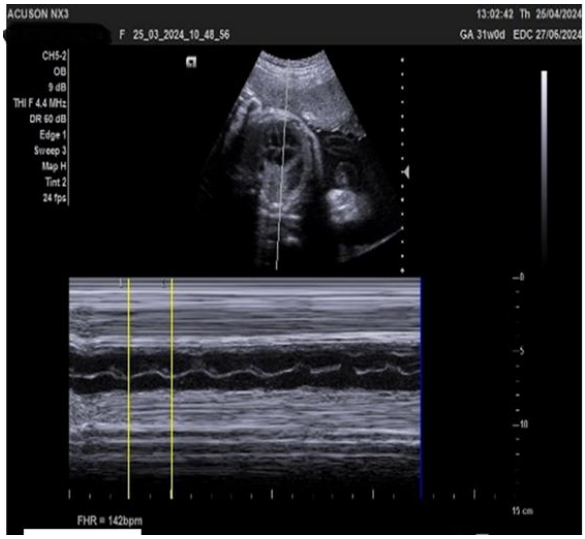


***Figure 3: Thermal images showing fetal heart rates at 31 weeks***

## Discussion

Fetal supraventricular tachycardia (SVT) is typically an isolated finding, with structural heart abnormalities present in up to 11% of cases.[3,4] In this case, detailed fetal echocardiography ruled out structural defects, negating the need for invasive genetic testing. The predominant cause of fetal SVT is atrioventricular reentrant tachycardia (AVRT), which was strongly suspected based on echocardiographic findings, though pulsed-wave Doppler (PWD) could have further confirmed the diagnosis. Sustained fetal tachyarrhythmia exceeding 200 bpm increases the risk of fetal heart failure and hydrops, with hydrops significantly worsening prognosis.[5,6] Early onset (<32 weeks) and persistent tachyarrhythmia further elevate this risk, necessitating prompt treatment.[5,7,8] Given the progression to hydrops at 24 weeks, transplacental therapy was initiated to prevent adverse outcomes.



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Digoxin is commonly the first-line treatment for fetal SVT, but its efficacy diminishes in hydropic fetuses.[9] Flecainide has demonstrated superior bioavailability and arrhythmia control, especially in hydropic conditions.[10-13] This case supports literature findings, as digoxin failed to resolve the tachycardia, but flecainide successfully restored sinus rhythm and reversed hydrops. Maternal tolerance to antiarrhythmics can impact treatment success. Despite comprehensive pre-treatment screening, the patient developed transient symptoms suggestive of drug intolerance at 32 weeks, necessitating dose adjustment. While maternal mirror syndrome and preeclampsia were considered, targeted evaluations ruled them out. Delivery timing remains variable, though most centers aim for 38 weeks. In this case, cesarean delivery at 37 weeks was chosen due to the absence of serum flecainide monitoring. Postnatal follow-up is crucial, as prolonged fetal arrhythmias and hydrops may affect neurodevelopment. This case highlights the effectiveness of transplacental flecainide therapy in fetal SVT with hydrops, aligning with existing evidence on its superiority in refractory cases.

## Conclusions

Early detection and intervention are crucial in managing fetal SVT, particularly when complicated by hydrops. This case demonstrates the effectiveness of transplacental flecainide therapy in achieving successful cardioversion, especially when first-line digoxin therapy fails. Careful maternal monitoring and dose adjustments are essential, especially in low-resource setting with no capacity for maternal serum flecainide monitoring. The case also reinforces the importance of prenatal evaluation, individualized management, and postnatal follow-up to optimize pregnancies complicated with fetal SVT in low-resource setting.

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